Aneurysmal degeneration and late rupture of an aortorenal vein graft: Case report, review of the literature, and implications for conduit selection

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The saphenous vein is among the most commonly used conduits for renal artery revascularization in adults. Vein grafts are more durable in the renal artery bed than in coronary and peripheral beds, and mechanisms of potential graft failure vary. Coronary vein grafts often fail because of atherosclerotic degeneration, whereas lower extremity grafts fail because of intimal hyperplasia or progression of underlying disease. In contrast, renal vein grafts may dilate over time but seldom fail. This may relate to the distinct hemodynamic environment of the renal bed with low-resistance, high-velocity blood flow. However, frank aneurysmal degeneration of renal vein grafts is rare with only a single report of rupture in the literature. We report an additional case of rupture of a late graft aneurysm and review the literature and our own experience with renal revascularization to underscore the rarity of this serious complication. The saphenous vein for aortorenal bypass grafting continues to be a favorable conduit for renal revascularization. Long-term duplex ultrasound scanning follow-up is recommended to survey the reconstructed artery and perhaps more important, to evaluate progression of subclinical disease in the contralateral renal artery. (J Vasc Surg 2000;32:612-5.)

CASE REPORT

A 75-year-old woman presented with severe hypertension, vague abdominal discomfort, and anemia 22 years after aortorenal vein graft repair of a critical right renal artery stenosis. She was referred to our center for further care and admitted for acute blood pressure control. Physical examination was remarkable for blood pressure of 200/100 mm Hg, mild abdominal tenderness in the right upper abdomen with a pulsatile mass and palpable thrill, and low-back tenderness but no flank pain or discoloration. Peripheral pulses were normal. Laboratory tests demonstrated a serum creatinine level of 1.2 mg/dL, hematocrit at 33%, and normal results from urinalysis. Further workup included renal duplex ultrasound scanning, which demonstrated a patent aortorenal vein graft with aneurysmal degeneration to a 6-cm maximum diameter. Abdominal computed tomography scan confirmed the vein graft aneurysm and demonstrated laminated mural thrombus (Fig 1). Selective angiography demonstrated a saccular defect in the graft wall proximal to the distal anastomosis (Fig 2).

After her blood pressure and cardiovascular status were optimized, the patient was taken to the operating suite for graft revision. During mobilization of the vein graft aneurysm, a posterior wall defect was encountered with a subacute, contained rupture proximal to the graft-renal artery anastomosis, which was intact (Fig 3, *A*). The aneurysm and the entire vein graft were resected, and the thrombus was evacuated. A sleeve endarterectomy of the aorta was performed, and a 6-mm polytetrafluoroethylene graft was interposed between the aorta and the native renal artery bifurcation (Fig 3, *B*). Intraoperative duplex scanning showed no intraluminal flow defects, and the postoperative course was uneventful. On discharge the patient was normotensive while taking two antihypertensive medications, and she had a serum creatinine level of 0.8 mg/dL. Serial duplex scanning graft surveillance was initiated.

DISCUSSION

The saphenous vein is the most widely used conduit for arterial bypass graft procedures; more than 1 million vein grafts are placed in the coronary and lower extremity vasculature in the United States

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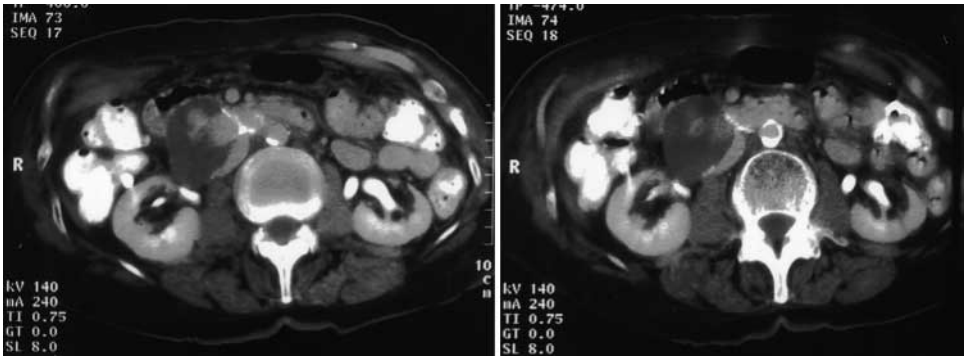

Fig 1. Abdominal computed tomography scan with intravenous contrast demonstrating right aortorenal vein graft aneurysm with laminated mural thrombus.

each year.[1] The vein is also the most common conduit used for renal artery reconstructions in adults. In coronary and peripheral arterial beds, vein graft failure is relatively common. Etiologies of failure include acute thrombosis, early intimal hyperplasia, and late atherosclerotic degeneration or progression of underlying arterial disease. The leading cause of graft failure in the coronary circulation is late graft atherosclerotic degeneration following characteristic "arterialization" and intimal thickening.[2,3] Lower extremity grafts have a higher incidence of early graft failure due to intimal hyperplasia or valve sclerosis and late failures due to progression of disease in the native vasculature. In contrast, failure of vein grafts in the renal arterial bed is uncommon. Causes most commonly include stenosis and thrombosis.[4,5]

Dilation of renal grafts has been described, but frank aneurysmal degeneration is unusual.[4,5] To our knowledge, rupture has only been reported once in the previous literature[6] with the current report contributing the second case, underscoring the rarity of this serious complication. Of the very large number of aortocoronary vein grafts performed with extensive late follow-up, only 31 true aneurysms have been described in the literature with an average time to presentation of 10.1 years.[7,8] Lower extremity vein grafts infrequently develop aneurysmal degeneration. Although these cases are probably underreported, in a recent review of the literature, 19 patients with 27 nonanastomotic aneurysms were described with an average time to presentation of 7.8 years.[9]

In 1973, Stanley et al[10] published the follow-up of 100 aortorenal vein bypass grafts and warned of the potential for aneurysmal degeneration of the saphenous vein when used in the pediatric population with renovascular hypertension. However, in a review of complications of renal artery reconstruction that required reoperation, aneurysm formation was extremely rare, accounting for only three (1%) of 278 aortorenal saphenous vein bypass grafts requiring revision.[4] The ages of the three patients were 15, 18, and 49 years, and the average time to revision was 28 months.[4] In a separate review of 198 renal bypass grafts followed with angiography, pathologic dilatation was noted in five saphenous vein grafts, and all were in children. All were followed by initial observation, and none required reoperation because the initial enlargement stabilized on serial follow-up examinations.[11] From 1987 to 1997, we performed more than 780 renal artery

Fig 2. Selective arteriogram demonstrating aneurysmal vein graft with saccular defect, which at operation was a subacute contained rupture. *Arrowheads* denote the proximal and distal anastomosis.

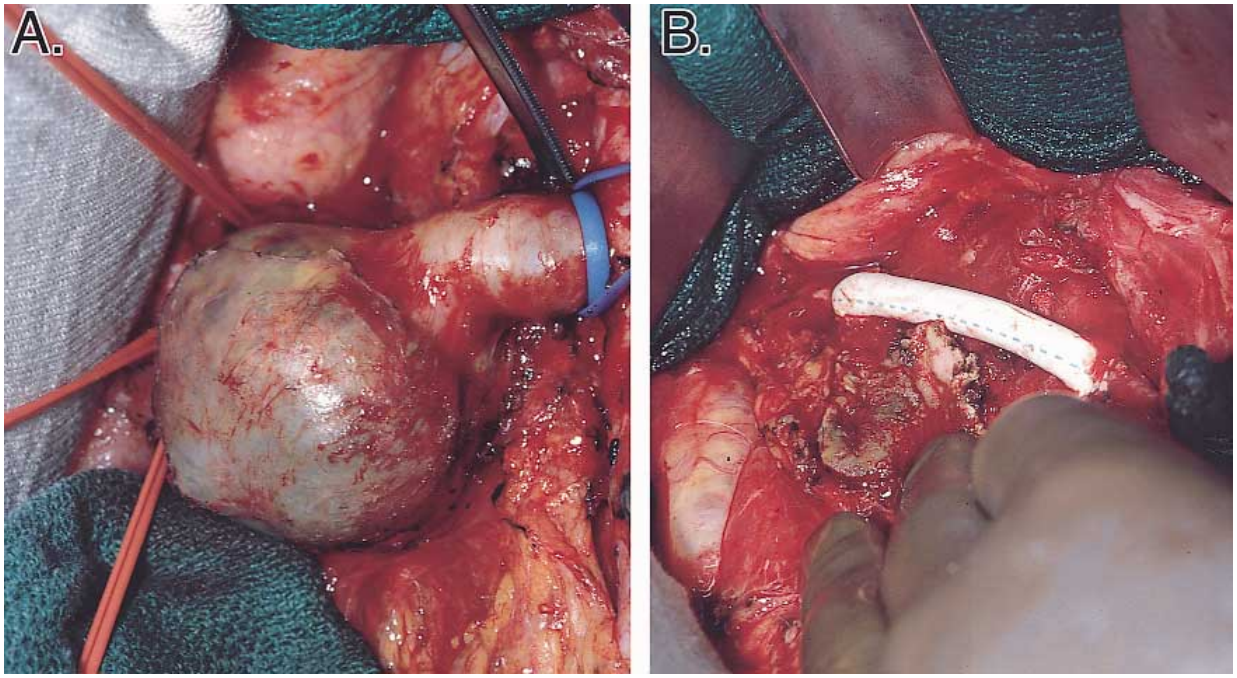

Fig 3. A, Intraoperative photograph of aortorenal vein graft aneurysm. B, After resection of the aneurysmal vein graft in its entirety, the renal artery was reconstructed with an aortorenal polytetrafluoroethylene interposition graft.

reconstructions of which 290 were saphenous vein grafts with a mean follow-up of 27 months. No vein grafts have required revision for dilatation, and none have ruptured.[5]

The cause of saphenous vein bypass graft enlargement with progression to aneurysm formation is unknown. The distinct hemodynamic environment of the renal bed with its low-resistance and high-velocity blood flow may lead to enlargement of vein grafts much as a radial-cephalic fistula matures. It has been suggested that the disruption of the vasa vasorum, especially in children in whom the adventitial and medial plexuses are considered immature, contributes to local ischemia and focal dilation.[12] It has been shown experimentally that vein grafts placed for arterial bypass grafting reestablish their nonluminally derived medial and adventitial blood supply within 48 to 72 hours.[13] Poststenotic dilatation has also been shown to lead to aneurysm formation in saphenous vein grafts, which further highlights the implications of hemodynamic disturbances. Although the true cause of these aneurysms remains to be elucidated, their incidence remains extremely low. This case represents only the second report of a ruptured aortorenal saphenous vein bypass graft and is the first in which a salvage bypass graft has been performed.[6]

In our view, the rarity of late complications serves to highlight the durability of the saphenous vein for use in renal revascularization. Long-term duplex ultrasound scanning follow-up is recommended to follow the reconstruction and perhaps more important, to evaluate progression of subclinical disease in the contralateral renal artery. Used for aortorenal bypass grafting, the saphenous vein continues to be equal to or surpasses synthetic materials, with respect to resistance to infection, ease of handling for branch artery repair, and primary patency. In our experience, the vein continues to be a favorable conduit for renal revascularization.

REFERENCES

1. American Heart Association. 2000 Heart and Stroke statistical update. Available from: www.americanheart.org/statistics/index.html. 2000.
2. Jones M, Conkle DM, Ferrans VJ, et al. Lesions observed in arterial autogenous vein grafts: light an electron microscopic evaluation. Circulation 1973;48 Suppl 3:198-210.
3. Motwani JG, Topol EJ. Aortocoronary saphenous vein graft disease: pathogenesis, predisposition, and prevention. Circulation 1998;97:916-31.
4. Stanley JC, Whitehouse WM, Zelenock GB, Graham LM, Cronenwett JL, Lindenauer SM. Reoperation for complications of renal artery reconstructive surgery undertaken for treatment of renovascular hypertension. J Vasc Surg 1985;2:133-44.

5. Hansen KJ, Deitch JS, Oskin TC, Ligush J, Craven TE, Dean RH. Renal artery repair: consequences of operative failures. Ann Surg 1998;227:678-90.
6. Lavigne JP, Keppenne V, Limet R. Late rupture of a saphenous vein aortorenal graft. J Vasc Surg 1999;29:722-3.
7. Kalimi R, Palazzo RS, Graver LM. Giant aneurysm of saphenous vein graft to coronary artery compressing the right atrium. Ann Thorac Surg 1999;68:1433-7.
8. Navia D, Vaccarino G, Vrancic M, Piccinini F, Iparraguirre E. Surgical treatment of an asymptomatic large coronary saphenous vein graft aneurysm. J Thorac Cardiovasc Surg 1999;118:1122-3.
9. Cassina PC, Hailemariam S, Schmid RA, Hauser M. Infrainguinal aneurysm formation in arterialized autologous saphenous vein grafts. J Vasc Surg 1998;28:944-8.
10. Stanley JC, Ernst CB, Fry WJ. Fate of 100 aortorenal vein grafts: characteristics of late graft expansion, aneurysmal dilatation, and stenosis. Surgery 1973;74:931-44.
11. Dean RH, Hansen KJ. Renovascular hypertension. In: Moore WS, editor. Vascular surgery: a comprehensive review. Philadelphia: WB Saunders; 1998. p. 537.
12. Short RHD. The vasa vasorum of the femoral vein. J Pathol Bact 1940;50:419-22.
13. Wyatt AP, Rothnie NG, Taylor GW. The vascularization of vein-grafts. Br J Surg 1964;51:378-81.

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